## Comment 2000

Response Section in Chapter 35 ➥

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From: Cameron Alston Cova <cameroncova@yahoo.com>
To: <mountainview@utah.gov>
Date: Thursday - January 24, 2008 11:01 PM
Subject: Utah Moms for Clean Air and Utah Physicians for a Healthy Environment Comments

Attached are the comments to the Mountain View Corridor DEIS prepared by Utah Moms for Clean A
Utah Physicians for a Healthy Environment.

Thank you for your consideration.

Cameron Cova
Utah Moms for Clean Air
Chair, Transportation Committee





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**Comments to the Mountain View Corridor**

**Draft Environmental Impact Statement**

**Submitted by:**

**Utah Moms for Clean Air**

**And**

**Utah Physicians for a Healthy Environment**

**Introduction**

Utah suffers from a serious air quality problem. During times of winter inversion, the Wasatch Front is among the ten most acutely polluted areas in the country. During the hot summers, ozone levels can also rise to alarming levels. The brown haze that hangs over us is more than an eyesore – it literally sickens those who live here, especially the most vulnerable in our society: children, pregnant women, fetuses, the elderly, and anyone with compromised health. This pollution is largely, though not entirely, the result of motor vehicles.

Against the backdrop of this air quality reality, Utah is also at a development crossroads. The state is growing rapidly – adding population faster than almost any other state in the country. We have large swaths of undeveloped land that are likely to be filled with residential, industrial, and corporate development. The Mountain View Corridor is a crucial piece of this development puzzle in the state. The Western valley is still in its nascent development period, and decisions made now will shape the development patterns for generations. We have the opportunity to shape this development in a way that is economically robust while also ensuring a high quality of life and safe and healthy air quality for all those living here.

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A well-designed transportation plan, with robust public transit and well-placed, correctly-sized roadways, will shape that development for the common good. On the other hand, poor transportation decisions made now will negatively affect generations of Utahns.

While our organizations recognize that the growing population base in the western edge of the valley will require increased transportation options, we believe that the preferred alternative presented in the Mountain View Corridor Draft Environmental Impact Statement (hereinafter referred to as "the DEIS") does not adequately balance the need for increased road capacity with the parallel, and we argue paramount, need for improved air quality and improved transportation alternatives. In addition, the preferred alignment of the road, along 5800 West, is particularly problematic due to the known and serious health risks to those who live and attend school near its path. Specifically, four schools are within 500 meters of the proposed freeway, a distance that has been shown to be associated with poor lung development; three of those schools would be within 250 yards of the freeway, a distance that has been shown to be associated with an increase in, among other diseases, childhood cancer. The DEIS inadequately analyzes and discloses these health effects to the public and decision makers, making it impossible for them to make informed decisions about the future health and welfare of the state.

Utah Moms for Clean Air and Utah Physicians for a Healthy Environment believe that two fundamental changes must be made to the preferred alternative presented in the DEIS in order to adequately protect the health and welfare of those living along the Wasatch Front, as well as those attending school near the proposed freeway:

1) The alignment of the freeway along 5800 West must be abandoned. This alignment places children attending school nearby the freeway at an unacceptable risk of

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developing serious and potentially life-long health problems, with a series of related health and economic costs. UDOT should consider other alignments for the road that do not pass nearby schools.

2) The plan must include robust public transportation earlier in the development cycle so transit is built at the same time, or preferably before, the roadway in order to improve air quality along the entire Wasatch Front.

**A) Alignment of the Proposed Freeway along 5800 West Places Schoolchildren at Risk.**

**1) What the plan calls for.**

The preferred alternative in the DEIS, a six to eight lane freeway running along 5800 West, lies within 500 meters of four schools: Hunter High School, Hunter Junior High School, Hillside Elementary and Whittier Elementary. Three of those schools are within 500 *feet* of the proposed freeway: Hunter High, Whittier and Hillside. For those children living in the school boundary area, these schools represent the facilities they will attend during their entire thirteen-year primary education career. For example, the children who start at Whittier Elementary proceed to Hunter Jr. High and then move to Hunter High School will spend an average of eight hours a day, five days a week, from the time they are five or six years old until they are seventeen or eighteen years old within 500 meters of the freeway. As discussed below, this proximity to the highway will place these children at an unacceptable risk of developing serious health problems.

**2) Why the preferred alternative presented in the DEIS fails.**

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**a. The proximity of the freeway to school children presents serious and unacceptable health effects which are not discussed or disclosed fully in the DEIS, in violation of NEPA.**

Protecting the health of Utahns, specifically those most vulnerable in our population, should be among the paramount concerns of the state. Unfortunately the DEIS presents as its preferred alternative a freeway alignment that will place school children at an unacceptable risk of health problems. In addition, the DEIS fails to adequately discuss or disclose those risks to the public.

The DEIS mentions in cursory fashion a handful of recent studies that show an association between proximity to freeways and harm to public health. (DEIS at 12-33 through 12-35). This analysis is clearly inadequate given the gravity of the issues at stake. WE have included, as Appendix A to these comments, a summary of published, peer-reviewed studies that conclude that exposure to exhaust fumes from vehicles on freeways is linked to a variety of diseases, illnesses, and lung development problems.

For example, a 2000 Denver study showed that children living within 250 yards of streets or highways with 20,000 vehicles per day are six times more likely to develop all types of cancer and eight times more likely to get leukemia.[1] A 2004 Italian study found similar results,

[1] Pearson, Wachtel; Robert L. Pearson, and Kristie Ebie. (2000). Distance-weighted traffic density in proximity to a home is a risk factor for leukemia and other childhood cancers. *Journal of Air and Waste Management Association* 50:175-180.

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concluding that childhood leukemia was almost four times higher for heavily exposed children compared to children who did not live near roadways.[2]

Other studies show clear correlation between traffic exhaust and asthma. For example, a study of 1,498 children in 13 schools found a positive relationship between school proximity to highways and asthma occurrence. Truck traffic intensity and the concentration of pollutants measured in schools were found to be significantly associated with chronic respiratory symptoms.[3]

Proximity to roadways has also been correlated with reduced lung function and development in children. In a very recent Lancet study, children from 12 southern California communities who lived within 500 meters of a freeway were found to have substantial deficits in respiratory volume and flow, compared with children who lived at least 1500 meters from a freeway. The study showed that both local exposure to freeways and regional air pollution had detrimental, and independent, effects on lung-function growth.[4]

Due to our geography-induced inversions, PM 2.5 pollution is of particular concern in Utah. PM 2.5 is widely agreed to be among the most health threatening types of emissions –

[2] Crosignani P ;Tittarelli A; Borgini A;Codazzi T; Rovelli A; Porro E; Contiero P; Bianchi N; Tagliabue G; Fissi R; Rossitto F; Berrino F. Childhood Leukemia and Road Traffic: A population-based Case-Control study. *International Journal of Cancer,* 2004, V108, N4 (FEB 10), P 596-599.

[3] Van Vliet, P., M. Knape, et al. (1997).Motor vehicle exhaust and chronic respiratory symptoms in children living near freeways. Environmental Research. 74(2): 122-32.

[4] Effect of exposure to traffic on lung development from 10 to 18 years of age: a cohort study. *The Lancet*, Volume 369, Issue 9561, Pages 571-577. W. Gauderman, H. Vora, R. McConnell, K. Berhane, F. Gilliland, D. Thomas, F. Lurmann, E. Avol, N. Kunzli, M. Jerrett.

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because of its tiny size, PM 2.5 can penetrate deeply into lung tissue and even pass into the bloodstream. The Federal Environmental Protection Agency published a Criteria Document for Particulate Matter summarizing a substantial number of peer-reviewed scientific studies that show a clear correlation between exposure to fine particulate matter (PM 2.5) and a number of serious health effects, including increased risk of cancer, fatal heart attacks, strokes, and respiratory diseases.[5] In fact, because particulate matter has been shown to be damaging to human health at even lower levels than previously thought, the EPA recently revised the PM 2.5 air quality standards. Given these findings, UDOT had an obligation to fully consider this evidence when estimating, and disclosing to the public, the adverse health effects of emissions from the highway.

In fact, in recognition of the serious health effects of locating a school and a freeway in proximity to one another, California recently passed a state law prohibiting any new school construction within 500 feet of a freeway. Although such a law does not yet exist in Utah, the DEIS should take into account the changing legal landscape with respect to school and freeway proximity, as more and more evidence accumulates that close proximity of those uses put human health at risk.

Providing convenient transportation cannot and should not trump protecting children from harm. The health risks of the alignment of the proposed freeway along 5800 West are real and must be given much greater weight in the DEIS so that the public and the decision-makers can understand the true costs of the road.

[5] Environmental Protection Agency, *Air Quality Criteria for Particulate Matter* (EPA/600/P-99/002aF, EPA/600/P-99/002bF)(2004)

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**b. The DEIS fails to adequately consider and disclose the air pollution that will result from the freeway.**

The DEIS fails to take the air quality of the immediate surrounding area of the freeway into proper account.

Specifically, the DEIS fails to include an adequate evaluation or analysis of the health impacts of fine particulate matter and air toxics from motor vehicle emissions that will result from the approval and construction of the proposed freeway. Because it is known that the proposed freeway, in its 5800 West alignment, would pass perilously close to four schools, this failure is unacceptable and, as explained below in section A.3.a (pages 8-10), unnecessary since methods exist that would allow the impacts of the freeway on nearby residents and schoolchildren to be measured.

A substantial number of peer-reviewed scientific studies show the serious negative health impacts of mobile-source fine particulate matter and air toxics emissions, especially on persons living or attending school near major roadways, as discussed above and in the appendices hereto. These studies support the argument that the analysis of particulate matter and air pollution in the DEIS is inadequate because it fails to evaluate the local impacts of fine particulate emissions and air toxics emissions, in violation of the requirements of the National Environmental Policy Act.

Given the seriousness of the health problems associated with proximity to freeways, as discussed in detail above, and the fact that once this freeway is built it will continue to have impacts for generations, it is unacceptable to us that the health effects of this freeway are not taken into greater account prior its authorization.

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Furthermore, the expectation in the DEIS that cleaner fuels and emissions regulations will have a large positive impact on the air pollution created by the project is overstated. The very recent (2008) California South Coast Air Quality Management District Multiple Air Toxics Exposure Study (MATES III) indicates that *despite reductions in vehicle emissions from cleaner fuels*, exposure to the concentration of air toxics near major transportation corridors is still associated with unacceptably high cancer risks. Any transportation strategy that relies only on future "cleaner" fuels to protect public health is likely to be inadequate. Any comfort derived from the prospect that cleaner fuels will reduce air toxics at some time in the future also ignores the medical realities that exposure to air toxics has a profoundly disproportionate impact on fetal and childhood development leading to a startling array of adult morbidities. For example, the EPA acknowledges that 50% of lifetime cancer risk is accumulated by the age of two. Toxicity of heavy metal exposure can be thousands of times greater during fetal development than even later on in childhood.

Those individuals exposed to heavy traffic exhaust early in life will not have their health consequences ameliorated by the introduction of cleaner fuels after they have passed those important stages in physical or neurological development. To rely on the strategy that the area will gradually become cleaner essentially sacrifices the health of thousands of children exposed during critical developmental years.

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**c. The DEIS does not consider the economic costs related to the health effects of the freeway.**

In addition to failing to properly address the health effects of the freeway, the DEIS also fails to account for the *economic costs* associated with those health effects. The DEIS assigns economic values to factors such as delays caused by traffic congestion and performs a

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cost/benefit analysis between the alternatives based on the amount of economic harm caused by hours spent stuck in traffic. The DEIS completely fails to account for the economic impact of the *health problems* likely to be associated with the freeway, however.

Although dollar figures could never adequately measure the true impact of health problems as they relate to the individuals harmed, their families, and their communities, it can still act as a crude way of accounting for the financial impact of vehicle exhaust pollution. The cost of health care is a huge burden at the personal, corporate, and government level. The costs associated with illness caused by pollution includes current and future medical bills, reduced longevity of the population, reduced productivity of workers, and increased insurance costs, to name a few specific measures. If rerouting the freeway away from schools can prevent health problems from developing in the school children in the planned path of the freeway, it is inappropriate to leave those economic considerations out of the decision-making process.

**3) What should be done?**

35.12.4A

**a. UDOT should Assess Health Impacts from Fine Particulate Matter and Mobile-Source Air Toxics, Particularly Where the Road Passes Nearby Schools.**

The DEIS fails to include a robust health impact assessment for pollutants that are known to have adverse health impacts, as required under NEPA. Such assessments must be made before any final decision can be made about the project.

The DEIS indicates that that the Mountain View Freeway will increase mobile-source air toxic ("MSAT") emissions in the study area by nine to twelve percent relative to a non-build

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alternative, due primarily to increased VMT for the build alternative. (DEIS at 12-36). However, the DEIS does not evaluate localized MSAT emissions and concentrations near the freeway. These emissions are especially likely to be of concern in the early years of operation of the road, when MSAT emissions can be expected to be at their highest levels (because older more polluting vehicles will still be on the road in greater numbers). UDOT states that it is unable to evaluate localized emission concentrations or health effects because of uncertainties in the MOBILE6.2 model and uncertainties surrounding the health effects of MSAT pollutants. (DEIS at 12-32). However, exposure and risk assessment tools exist that UDOT could use to establish the degree of risk roadside populations would face from exposure to fine particulate matter, MSAT emissions and diesel particulate emissions.

35.12.4F

Specifically, two different methods are available to assess the risks to human health from particulate matter and mobile-source air toxics. Both methods combine estimates of exposure with estimates of the "dose-response" function (an estimate of the risk of a specific health effect in response to a specified exposure to the pollutant) to produce an estimate of risk associated with that exposure. One method is based on epidemiologic data that establishes how the risk of particular health effects changes with exposure to particular pollutants. The second method assesses cancer risks from exposure to MSATs as a result of the freeway by estimating changes in the concentrations of the six priority MSATs using EPA's MOBILE6.2 model. Both of these methods, as well as examples of where and how they have been used, are discussed at length in Dr. John Balbus April 2006 Statement, Appendix B.

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**b. UDOT should address the economic costs related to health impacts.**

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As discussed above, the DEIS should consider the economic costs associated with the negative health effects of the freeway generally, and specifically the costs associated with the health effects of its proposed alignment near the schools. Considering the economic benefit of reduced traffic delays and congestion without comparing those benefits with the economic costs of the health impacts fails to reveal the true economic reality of the planned highway, and makes a true, holistic comparison of the alternatives impossible.

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**c. UDOT should align freeway along 7200 West or consider other alternatives.**

If the road must be authorized before determining what the health effects of that choice will be, at the very least the alignment of the freeway must be shifted to reduce the known impact of the vehicle exhaust on children. For this reason, *of the alternatives included in the DEIS*, the 7200 West alternative is preferable to the 5800 West alignment because it does not pass in such close proximity to schools. Also, as indicated in the DEIS, the 7200 West alternative produces slightly less MSATs and particulate matter than the 5800 West alternative, which, in and of itself, argues for shifting the alignment away from 5800 West. However, we argue that because the health impacts include both the acute response to local pollution and the overall detriment to the health of people living all along the Wasatch Front, any other alternative alignment or transportation development plan that reduces the negative health effects of traffic exhaust also must be considered by UDOT before settling on a final road building plan.

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**B) Public Transportation Sequencing is Flawed**

**1) What the plan calls for.**

Although the DEIS does set forth a plan for public transportation along 5600 West, the sequencing of the implementation of that transit system comes far too late to have a positive impact on Utah's serious air quality problems. The Wasatch Front Regional Plan does not include any transit along 5600 West before the year 2030. Delaying transit until that time will have unacceptable long-term negative impacts on the transit ridership of the western valley.

The DEIS sequencing analysis states that there is no positive impact associated with building transit first. This statement is based largely on the opinions of currently-in-office elected officials in the municipalities along the corridor as to what their expectations are for development in their areas. This is an inadequate and flawed analysis. Awareness of the regional air quality problem has grown significantly in the last few years. The media has ramped up its reporting of the problem and grassroots groups such as ours are getting more and more involved with the issue; as a result, the public is becoming more interested in protecting our "air shed" and protecting the children and others who live here from the harmful effects of air pollution.

Citizen demand for public transit is growing every day and what may seem to be out of reach now, may very well be considered an obvious choice in the future. Likewise, awareness of planning in a way that encourages public transit ridership has been developing over time. As one recent example of the shifting attitude toward public transportation, in November 2006 Utah voters overwhelmingly voted to raise their own taxes to add TRAX lines in Salt Lake County

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and finish building the FrontRunner commuter rail from Ogden to Provo. This vote clearly demonstrates the public's commitment to mass transit. This reality is not reflected in the DEIS. Furthermore, the lack of transit-first approach also goes against the advice of the Governor's Blue Ribbon Advisory Council whose October 2007 report urged an aggressive mass transit strategy.

**2) Why the plan fails.**

**a. Air Quality problem in Utah is serious and requires immediate action.**

On certain days, Utah's air quality is among the worst in the country for acute spikes of pollution. PM 2.5 pollution is of particular concern here, and Salt Lake County will be in non-attainment for this pollutant under newly revised EPA guidelines. Awareness of the seriousness of the problem has grown and the question now is not whether there is a problem, but what can we do to improve it as soon as possible.

**b. Public transit decisions will drive development patterns and driving habits.**

Properly planned public transportation can shape the way the entire western side of the valley is developed. If robust public transportation (preferably light rail that connects to the current Trax system) is put into place, development will be oriented to take advantage of that public transportation.

**c. What should be in the plan?**

Utah Moms for Clean Air and Utah Physicians for a Healthy Environment believe that robust public transit should be built at the same time as, or preferably before the proposed road.

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Specifically, a fully integrated, center-running light rail system that connects with Trax, we believe, will have the greatest positive impact on local and regional air quality. Light rail has proven to be a huge success in Utah, with rider statistics far outpacing the forecasts predicted by the modeling preceding the projects. If the transit system is built before the freeway, or at the very latest at the same time as the freeway, development and driving patterns in the area will be fundamentally altered in a way that protects our air shed (despite the DEIS assumptions to the contrary). Simply put, under a transit-first approach, fewer people will drive and more people will ride public transit – the exact recipe Utah needs to address our critical air pollution problems.

**Conclusion**

Utah has the opportunity to make transportation decisions in the undeveloped western edge of the valley that will impact generations of its residents. If planned well, the transportation can serve the needs of those living, working and traveling in and out of that section of the state while also preserving and protecting the health of our most vulnerable communities. If planned poorly, thousands of Utahns will literally suffer the consequences.

New health studies summarized in these comments show that emissions from a project carrying over 100,000 vehicles per day, as the Mountain View Corridor is expected to do, will be associated with childhood cancer, impairment of children's lung development, increased incidents of asthma and other allergy-related immune conditions in children among other health problems for those living or attending school within 500 meters of the freeway. Adults will be at greater risk of cardiovascular disease, and the elderly living in the corridor will experience greater respiratory disease. This evidence triggers the obligation to reconsider the freeway along

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with other alternatives to avoid or minimize these effects, and to determine whether the highway is in the best overall public interest.

Likewise, due to the overall air pollution problems in the state, a robust public transit strategy is needed. Simply allowing the Mountain View Corridor region to develop around a freeway would condemn our future to continued poor air quality. Public transportation added into the mix as an afterthought would be hobbled in its ability to truly provide for the region's transportation needs.

**Respectfully Submitted:**

**Utah Moms for Clean Air**

**and**

**Utah Physicians for a Healthy Environment**

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APPENDIX A

In this appendix we summarize the studies showing the link between traffic-related pollution and health risks. The medical data regarding increased public health risks to residents who live within 500 to 1500 ft. of a major roadway and especially one with heavy diesel traffic is very clear. The use of indicator pollutants to assess exposures is appropriate but should not be interpreted as demonstrating that observed health effects are related only to exposures to the indicators. It is plausible if not likely that exposure to the complex mixture of traffic-related pollution is more harmful than exposure to only one or two of the primary constituents of the mixture.

The studies also suggest that health risks are elevated at traffic counts in the thousands and low tens of thousands of vehicles per day, far below the anticipated traffic on the Mountain View Corridor freeway. Strengths of the new studies include the fact that several involve following cohorts of children over time, which provides more certainly in the diagnosis of asthma and other conditions.

The most alarming of the studies mentioned here are those that indicate extremely high concentrations of ultrafine particulate matter near freeways, the growing understanding of the uniquely toxic effects of ultrafines and the just released data on intrauterine growth retardation from only modest traffic related air pollution. Taken together all these studies strongly suggest that UDOT must more closely assess the true health impact of the Mountain View Corridor on those most likely to be affected.

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**Studies Supporting the Association between Air Pollution and Cancer**

**Children Living Near Busy Roads More Likely to Develop Leukemia and Cancer**

A 2000 Denver study showed that children living within 250 yards of streets or highways with 20,000 vehicles per day are six times more likely to develop all types of cancer and eight times more likely to get leukemia. The study looked at associations between traffic density, power lines, and all childhood cancers with measurements obtained in 1979 and 1990. It found a weak association from power lines, but a strong association with highways. It suggested that Volatile Organic Compound pollution from traffic may be the cancer promoter causing the problem.

Pearson, Wachtel; Robert L. Pearson, and Kristie Ebie. (2000). Distance-weighted traffic density in proximity to a home is a risk factor for leukemia and other childhood cancers. *Journal of Air and Waste Management Association* 50:175-180.

**Road Traffic Contributes to the Origin of Childhood Leukemia**

A 2004 Italian study found that childhood leukemia is partially caused by roadside emissions in the Province of Varese. The authors conducted a population-based, case-controlled study in the Province of Varese, northern Italy, which was covered by a population-based cancer registry. Their study found that the risk of childhood leukemia was almost four times higher for heavily exposed children compared to children whose homes were not exposed to road traffic emissions of benzene. Children either inhale benzene as a gas or particulate matter which has absorbed benzene. Their model included traffic density divided into two groups–one greater and one less than 10,000 vehicles per day, distance, and weather conditions to estimate benzene concentration. The researchers' data suggests that motor vehicle traffic emissions are involved in the origin of childhood leukemia.

"Childhood Leukemia and Road Traffic: A population-based Case-Control study."Crosignani P ;Tittarelli A; Borgini A;Codazzi T; Rovelli A; Porro E; Contiero P; Bianchi N; Tagliabue G; Fiasi R; Rosatto F; Berrino F. *International Journal of Cancer,* 2004, V108, N4 (FEB 10), P 596-599.

**Exposure to Cancer-Causing Benzene Higher for Children Living Near High Traffic Areas**

German researchers compared 48 children who lived in a central urban area with high traffic density with 72 children who lived in a small city with low traffic density. They found that the blood levels of benzene in children who lived in the high-traffic-density area were 71 percent higher than those of children who lived in the low-traffic-density area. Blood levels of toluene and carboxyhemoglobin (formed after breathing carbon monoxide) were also significantly elevated (56 percent and 33 percent higher, respectively) among children regularly exposed to vehicle pollution. Aplastic anemia, a serious condition in which bone marrow stops producing blood cells, and leukemia were associated with excessive exposure to benzene.

Jermann E, H. Hajimiragha, A. Brockhaus, I Freier, U. Ewers, A. Roscovanu: Exposure of children to benzene and other motor vehicle emissions. Zentralblatt fur Hygiene und Umweltmedizin 189:50-61, 1989.

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**Motor Vehicle Exhaust Dominates Cancer Risk from Air Pollution**

The most comprehensive study of urban toxic air pollution ever undertaken shows that motor vehicles and other mobile sources of air pollution are the predominant source of cancer-causing air pollutants in Southern California. Overall, the study showed that motor vehicles and other mobile sources accounted for about 90 percent of the cancer risk from toxic air pollution, most of which is from diesel soot (70 percent of the cancer risk). Industries and other stationary sources accounted for the remaining 10 percent. The study showed that the highest risk is in urban areas where there is heavy traffic and high concentrations of population and industry.

South Coast Air Quality Management District. Multiple Air Toxics Exposure Study-II.March 2000.

**Traffic Related Cancer Risk Still High After Introduction of Cleaner Fuels**

In a follow up to the above mentioned study, the California South Coast Air Quality Management District essentially repeated the study during a period of six to eight years later after cleaner fuels had been introduced to the marketplace. They found that mobile sources still represented 94% of the overall air toxic related cancer risk with diesel exhaust being the major component of that risk. Levels of air toxics both monitored and modeled showed varying reductions in concentration from the first study to the second, and the overall cancer risk had dropped by 17% to 1.2 per thousand. Nonetheless, the cancer risks were still unacceptably high and highest in the vicinity of transportation corridors.

South Coast Air Quality Management District. Multiple Air Toxics Exposure Study-III. March 2008. Currently in draft form.

**Cancer Risk Higher Near Major Sources of Air Pollution, Including Highways**

A 1997 English study found a cancer corridor within three miles of highways, airports, power plants, and other major polluters. The study examined children who died of leukemia or other cancers from the years 1953-1980, where they were born and where they died. It found that the greatest danger lies a few hundred yards from a highway or polluting facility and decreases as you get further away from the facility.

Knox and Gilman (1997). Hazard proximities of childhood cancers in Great Britain from 1953-1980. *Journal of Epidemiology and Community Health.* 51: 151-159.

**Soot Particulate Matter Linked to Lung Cancer and Cardiopulmonary Mortality**

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A recent study appearing in the Journal of the American Medical Association showed that day-to-day exposure to soot or fine particulate matter, a major component of tailpipe pollution increased the risk of various adverse health effects. More specifically

the study shows that each 10 microgram/$m^3$ elevation in fine particulate air pollution leads to an 8 percent increased risk of lung cancer deaths, a 6 percent increased risk of cardiopulmonary mortality (heart attacks and strokes) and 4 percent increased risk of death from general causes.

Pope, Clive Arden III; Richard P. Burnett, et al. Lung Cancer, Cardiopulmonary Mortality, and Long-term Exposure to Fine Particulate Air Pollution. *Journal of the American Medical Association*, March 6 2002— Vol. 287, No. 92.

**Studies Supporting the Association between Vehicle Exhaust and Asthma**

**Increasing Public Transportation and Cutting Traffic Reduces Asthma Attacks**

This 2001 Journal of the American Medical Association study found that increasing public transportation along with other traffic control measures during the 1996 Atlanta Olympics reduced acute asthma attacks by up to 44 percent in children, reduced ozone concentrations by 28 percent, and morning peak traffic by 22.5 percent. These data provide support for efforts to reduce air pollution and improve health via reductions in motor vehicle traffic.

Friedman, Michael; Kenneth Powell MD; Lori Hutwagner; Leroy Graham; Gerald Teague. Impact of Changes in Transportation and Commuting Behaviors During the 1996 Summer Olympic Games in Atlanta on Air Quality and Childhood Asthma, *Journal of the American Medical Association*, 2001; 285:897-905.

**Truck Traffic Linked to Childhood Asthma Hospitalizations**

A study in Erie County, New York (excluding the city of Buffalo) found that children living in neighborhoods with heavy truck traffic within 220 yards of their homes had increased risks of asthma hospitalization. The study examined hospital admission for asthma amongst children ages 0-14, and residential proximity to roads with heavy traffic. Lin, Shao;

Jean Pierre Munsie; Syni-An Hwang; Edward Fitzgerald; and Michael R. Cayo; (2002). Childhood Asthma Hospitalization and Residential Exposure to State Route Traffic. Environmental Research, Section A, Vol. 88, pp. 73-81.

**Traffic-Related Air Pollution Associated with Respiratory Symptoms in Two Year Old Children**

This cohort study in the Netherlands found that two year old children who are exposed to higher levels of traffic-related air pollution are more likely to have self-reported respiratory illnesses, including wheezing, ear/nose/throat infections, and reporting of physician-diagnosed asthma, flu or serious cold.

Brauer, Dr.Michael J. et al. (2002). Air Pollution from Traffic and the Development of Respiratory Infections and Asthmatic and Allergic Symptoms in Children. *American Journal of Respiratory and Critical Care Medicine*. Vol. 166 pp 1092-1098.

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### Asthma Symptoms Caused by Truck Exhaust

A study was conducted in Munster, Germany to determine the relationship between truck traffic and asthma symptoms. In total, 3,703 German students, between the ages of 12-15 years, completed a written and video questionnaire in 1994-1995. Positive associations between both wheezing and allergic rhinitis and truck traffic were found during a 12 month period. Potentially confounding variables, including indicators of socio-economic status, smoking, etc., did not alter the associations substantially.

Duhme, H.; S.K.Weiland, et al. (1996).The association between self-reported symptoms of asthma and allergic rhinitis and self-reported traffic density on street of residence in adolescents. Epidemiology 7(6):578-82.

### Proximity of a Child's Residence to Major Roads Linked to Hospital Admissions for Asthma

A study in Birmingham, United Kingdom, determined that living near major roads was associated with the risk of hospital admission for asthma in children younger than five years of age. The area of residence and traffic flow patterns were compared for children admitted to the hospital for asthma, children admitted for non-respiratory reasons, and a random sample of children from the community. Children admitted with an asthma diagnosis were significantly more likely to live in an area with high traffic flow (more than 24,000 vehicles/ 24 hrs) located along the nearest segment of main road.

Edwards, J.; S.Walters, et al. (1994). Hospital admissions for asthma in preschool children: relationship to major roads in Birmingham, United Kingdom. Archives of Environmental Health. 49(4): 223-7.

### Asthma More Common for Children Living Near Highways

A study of nearly 10,000 children in England found that wheezing illness, including asthma, was more likely with increasing proximity of a child's home to main roads. The risk was greatest for children living within 90 yards of the road.

Venn et al. (2001). Living Near A Main Road and the Risk of Wheezing Illness in Children. *American Journal of Respiratory and Critical Care Medicine.* Vol. 164, pp 2177-2180.

### Exposure to Nitrogen Dioxide (NO2) from Vehicles Exacerbates Asthma Attacks

Researchers at St. Mary's Hospital in Portsmouth, England determined that while 80 percent of asthma attacks are initially caused by viral infections, exposure to traffic pollution can increase symptoms as much as 200 percent. The team measured the exposure of 114 asthmatic children between ages eight-eleven from nonsmoking families over almost a whole year. They found a strong correlation between higher NO2 pollution and the severity of an attack.

Chauhan, A.J., et al. Personal exposure to nitrogen dioxide (NO2) and the severity of virus-induced asthma in children. *Lancet.* Volume 361 Issue 9373 Page 1939.

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## Comment 2000 (continued)

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### A School's Proximity to Highways Associated with Asthma Prevalence

A study of 1,498 children in 13 schools in the Province of South Holland found a positive relationship between school proximity to highways and asthma occurrence. Truck traffic intensity and the concentration of pollutants measured in schools were found to be significantly associated with chronic respiratory symptoms.

Van Vliet, P., M. Knape, et al. (1997).Motor vehicle exhaust and chronic respiratory symptoms in children living near freeways. Environmental Research. 74(2): 122-32.

### Diesel Exhaust Linked to Asthma

This study found that particulate matter from diesel trucks can act as an irritant in the airway causing asthma. The authors show that diesel exhaust can trigger asthma attacks in individuals with no pre-existing asthmatic history. When a natural allergen, such as pollen, was added to the situation, the reaction was even more dramatic.

Pandya, Robert, et al. "Diesel Exhaust and Asthma: Hypothesis and Molecular Mechanisms of Action." Environmental Health Perspectives Supplements Volume 110, Number 1, February 2002.

### Low Levels of Air Pollution Cause Asthma Attacks

Exposure to miniscule amounts of ozone and soot particulate matter 2.5 µm or less (PM2.5) in air at levels above current U.S. Environmental Protection Agency (EPA) standards is a risk factor for respiratory symptoms in children with asthma. Daily respiratory symptoms and medication use were examined prospectively for 271 children younger than 12 years with physician-diagnosed, active asthma residing in southern New England. Exposure to ambient concentrations of ozone and PM 2.5 from April 1 through September 30, 2001, was assessed using ozone (peak 1-hour and 8-hour) and 24-hour PM 2.5. Logistic regression analyses using generalized estimating equations were performed separately for maintenance medication users (n = 130) and nonusers (n = 141). Associations between pollutants (adjusted for temperature, controlling for same- and previous-day levels) and respiratory symptoms and use of rescue medication were evaluated. Mean (SD) levels were 59 (19) ppb (one-hour average) and 51 (16) ppb (8-hour average) for ozone and 13 (8) µg/m3 for PM2.5. In co-pollutant models, ozone level but not PM2.5 was significantly associated with respiratory symptoms and rescue medication use among children using maintenance medication; a 50-ppb increase in one-hour ozone was associated with increased likelihood of wheeze (by 35 percent) and chest tightness (by 47 percent).The highest levels of ozone (one-hour or eight-hour averages) were associated with increased shortness of breath and rescue medication use. No significant, exposure-dependent associations were observed for any outcome by any pollutant among children who did not use maintenance medication. Asthmatic children using maintenance medication are particularly vulnerable to ozone, controlling for exposure to fine particles, at levels below EPA standards.

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Gent, Janneane PhD; Elizabeth W.Triche, PhD; Theodore R. Holford, PhD; Kathleen Belanger, PhD; Michael B. Bracken, PhD; William S. Beckett,MD;Brian P. Leaderer,PhD,Association of Low-Level Ozone and Fine Particles With Respiratory Symptoms in Children With Asthma, *Journal of the American Medical Association.* 2003; 290:1859-1867.

**Studies Supporting the Association between Air Pollution and Prematurity, Low Birth Weight and Intrauterine Growth Retardation**

**Pregnant Women Who Live Near High Traffic Areas More Likely to Have Premature and Low Birth Weight Babies**

Researchers observed an approximately 10-20 percent increase in the risk of premature birth and low birth weight for infants born to women living near high traffic areas in Los Angeles County. In particular, the researchers found that for each one part-per-million increase in annual average carbon monoxide concentrations where the women lived, there was a 19 percent and 11 percent increase in risk for low-birth weight and premature births, respectively.

Wilhelm, Michelle and Beate Ritz. (2002). Residential Proximity to Traffic and Adverse Birth Outcomes in Los Angeles County, California, 1994-1996. *Environmental Health Perspectives.* doi: 10.1289/ehp.5688.

**Intrauterine growth retardation associated with traffic exhaust**

In one of the most important studies done to date on the impacts of air pollution, researchers in Australia found a significant correlation between exposure to primarily traffic exhaust air pollution and a reduction in fetus size. The lead author said, "The study found that mothers with a higher exposure to air pollution and fetuses that were, on average, smaller in terms of abdominal circumference, head circumference and femur length. Birth weight is a major predictor of later health." For example, bigger babies have been shown to have higher IQs in childhood and lower risk of cardiovascular disease, diabetes, and cancer and even obesity in adulthood.

*Environmental Health Perspectives,* A. Barnett , C. Hansen

**Studies Supporting the Association between Air Pollution and Stunted Lung Development**

**Children Living Near Highways Suffer Pronounced Deficits in Lung Function**

In this prospective study of 3677 children from 12 southern California communities who lived within 500 meters of a freeway had substantial deficits in 8-year growth of forced expiratory volume in 1 second (FEV1) and maximum midexpiratory flow rate (MMEF), compared with children who lived at least 1500

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## Comment 2000 (continued)

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meters from a freeway. Joint models showed that both local exposure to freeways and regional air pollution had detrimental, and independent, effects on lung-function growth.

Effect of exposure to traffic on lung development from 10 to 18 years of age: a cohort study. *The Lancet*, Volume 369, Issue 9561, Pages 571-577. W. Gauderman, H. Vora, R. McConnell, K. Berhane, F. Gilliland, D. Thomas, F. Lurmann, E. Avol, N. Kunzli, M. Jerrett.

**Lung Function Reduced Among Children Living Near Truck Traffic**

A European study determined that exposure to traffic-related air pollution, "'in particular diesel exhaust particles," may lead to reduced lung function in children living near major motorways.

Brunekreef, B; N.A. Janssen ; J. DeHartog; H. Harssema ;M. Knape; P. Van Vliet (1997). "Air pollution from truck traffic and lung function in children living near motorways." Epidemiology. 8(3):298-303.

**Other Important Studies**

**People Who Live Near Freeways Exposed to 25 times more Ultrafine Particulate Pollution**

Studies conducted in the vicinity of Interstates 405 and 710 in Southern California found that the number of ultra-fine soot particles in the air was approximately 25 times more concentrated near the highways and that pollution levels gradually decrease back to normal (background) levels around 300 meters, or nearly 330 yards, downwind from the highway. The researchers note that motor vehicles are the most significant source of ultra-fine particles, which have been linked to increases in mortality and morbidity. Recent research concludes that ultra-fine soot particles are more toxic than larger particles with the same chemical composition. Moreover, the researchers found considerably higher concentrations of carbon monoxide pollution near the highways.

Zhu, Yifang; William C. Hinds; Kim Seongheon; Si Shen; Constantinos Sioutas. Concentration and size distribution of ultrafine particles near a major highway. *Journal of the Air and Waste Management Association.* September 2002. And, Study of ultrafine particles near a major highway with heavy-duty diesel traffic. Atmospheric Environment. 36(2002), 4323-4335.

**Ultrafine Particulate Matter found to be much more toxic than larger particulate matter**

Ambient ultrafine particles (UFP)s defined as those with a diameter of less than .18um are by far the most abundant particles by number in the urban environment. Many recent studies have suggested that they are the most dangerous part of particulate matter because they can penetrate the most deeply into the lungs and can even be translocated directly from the nose into the brain. Researchers demonstrated in laboratory animals that UFP was far more potent than PM2.5 in promoting a cascade of inflammation and oxidative stress that resulted in the development of atherosclerosis. The development was demonstrated after only 75 hours of exposure spread over a period of 40 days.

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## Comment 2000 (continued)

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J. Araujo, B. Barajas, M, Kleinman. Ambient Particulate Pollutants in the Ultrafine Range Promote Early Atherosclerosis and Systemic Oxidative Stress. *Circulation Research*, Jan. 17, 08 online version.

**Air Pollution from Busy Roads Linked to Shorter Life Spans for Nearby Residents**

Dutch researchers looked at the effects of long-term exposure to traffic-related air pollutants on 5,000 adults. They found that people who lived near a main road were almost twice as likely to die from heart or lung disease and 1.4 times as likely to die from any premature cause compared with those who lived in less-trafficked areas. The authors say traffic emissions contain many pollutants that might be responsible for the health risks, such as ultra-fine particles, diesel soot, and nitrogen oxides, which have been linked to cardiovascular and respiratory problems.

Hoek, Brunekreef, Goldbohn, Fischer, van den Brandt (2002).Association Between Mortality and Indicators of Traffic-related Air Pollution in the Netherlands: A Cohort Study. *Lancet*, 360 (9341):1203-9.

**Five Times More Deaths Due to Air Pollution than Traffic Accidents**

This study analyzed the affect of traffic-related air pollution and traffic accidents on life expectancy in the area of Baden-Wurttemberg, Germany. It estimated that almost five times more deaths in this region resulted from motor vehicle pollution than from traffic accidents.

Szagun and Seidel. (2000). Mortality due to road traffic in Baden-Aarttemberg. Gesundheitswesen. 62(4): 225-33.

**Motor Vehicle Air Toxins Cause High Pollution Levels Inside Homes**

An air pollution study was done as a part of the West Oakland Diesel Truck Emissions Reduction Initiative. Researchers measured diesel particulates near mobile and idling trucks at the West Oakland Port. An aethalometer was used to measure indoor toxins and a high level of diesel particulates was found. The people who lived in these homes were exposed indoors to five times the level of diesel particulates that people were exposed to outdoors in other areas of Oakland.

W. Buchan, M.D. and M. Chan Jackson; Container Truck Traffic Assessment and Potential Mitigation Measures for the West Oakland Diesel Truck Emission Reduction Initiative, from "Clearing the Air, Reducing Diesel Pollution in West Oakland," a Report to Pacific Institute, 654 13th Street, Preservation Park, Oakland, California 94612, by TIAX LLC, 1601 S. De Anza Blvd., Suite 100, Cupertino, California 95014, November, 2003

**Regulators are underestimating traffic pollution**

Professors of chemistry and chemical engineering from Carnegie Mellon University reported in the esteemed journal *Science,* a new conceptual model for how microscopic particles behave in the atmosphere that suggest current air quality standards are inadequate. The authors state that their results indicate government officials need to adopt new ways of measuring and regulating fine particulate matter.

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## Comment 2000 (continued)

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They found new chemical processes that occur after soot and gaseous pollutants are emitted from vehicles, changing the chemical and physical properties of the particles and resulting in the creation of new particulate matter. These new particles are likely to be more toxic. Furthermore, these chemical processes lead to a spreading of pollution over a larger geographic area and help explain why urban air pollution can spread much further than previously thought.

Robinson AL, Donahue NM, Shrivastava MK, Weitkamp EA, Sage AM, Grieshop AP, Lane TE, Pierce JR, Pandis SN. Rethinking organic aerosols: semivolatile emissions and photochemical aging. *Science* 2007 Mar 2;315(5816):1259-62

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## Comment 2000 (continued)

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APPENDIX B

The following documents, which summarize health studies related to traffic pollution and human health, were prepared by Dr. John Balbus, the Director of the Health Program at Environmental Defense, an environmental non-profit group. The statements were prepared in reference to a highway project recently under consideration in another state. Although the road under consideration was not the Mountain View Corridor, the information is clearly relevant to this DEIS and should be considered by UDOT.

## Comment 2000 (continued)

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Statement of Dr. John Balbus

Director, Health Program

Environmental Defense

April 10, 2006

**I. Introduction**

This statement concerns the failure of the Federal Highway Administration and Maryland State Highway Administration to prepare an adequate project level conformity analysis of PM 2.5 or an adequate environmental impact statement ("EIS") to evaluate the health impacts of fine particulate matter and air toxics from motor vehicle emissions that will result from the approval and construction of the proposed Intercounty Connector in Maryland.

In this statement, I demonstrate that there exists a substantial body of peer-reviewed scientific studies showing the deleterious health impacts of mobile source fine particulate matter and air toxics emissions, especially on persons living or attending school near major roadways. These scientific studies support the argument that the final Environmental Impact Statement and proposed project level conformity analysis for fine particulates for the Intercounty Connector are inadequate because they fail to evaluate the local project hot spot impacts of fine particulate emissions and air toxics emissions, in violation of the requirements of the National Environmental Policy Act.

**II. Peer-Reviewed Scientific Studies Demonstrate That Motor Vehicle Emissions Of Fine Particulate Matter And Air Toxics Have A Significant Impact On Health.**

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## Comment 2000 (continued)

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Motor vehicles emit a soup of fine particulate matter ("$PM_{2.5}$")[6] and toxic air pollutants, and motor vehicle emissions are a substantial source of these pollutants in the ambient air.[7] The most recent EPA Criteria Document for Particulate Matter summarizes a substantial number of peer-reviewed, scientific studies that find a clear correlation between fine particulate matter and numerous health effects, including increased risk of fatal heart attacks, strokes, respiratory disease, and cancer.[8] Since 2000, published scientific studies show that mobile source air toxics include six known or suspected carcinogens, as well as respiratory irritants that may trigger asthma attacks.[9] Recent epidemiologic studies demonstrate the serious health consequences from exposure to mobile source air toxics and fine particulate matter. Methodological tools are available to evaluate the health risks from exposure to particulate matter and mobile source air toxics from traffic in close proximity to the proposed Intercounty Connector.

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[6] $PM_{2.5}$ refers to particulate matter less than 2.5 microns.

[7] See, e.g., Brauer, et al., *Estimating Long-Term Average Particulate Air Pollution Concentrations: Application of Traffic Indicators and Geographic Information Systems*, 14(2) Epidemiology 228 (2003) (a study conducted in three sites in Europe demonstrated that most of the variability in annual average concentrations of fine particulate matter was explained by vehicular traffic); Environmental Protection Agency, *Technical Support Document: Control of Emissions of Hazardous Air Pollutants from Motor Vehicles and Motor Vehicle Fuels*, EPA420-R-00-023, Table IV, A-1, p. 81 (2000) (EPA study estimating that motor vehicles accounted for 48% of the national total of benzene emissions, 43% of 1, 3-butadiene, 29% of acetaldehyde, and 24% of formaldehyde). Because air monitors generally do not collect data near roadways, these studies downplay the significance of human exposures to mobile source emissions near roadways.

[8] Environmental Protection Agency, *Air Quality Criteria for Particulate Matter* (EPA/600/P-99/002aF, EPA/600/P-99/002bF)(2004)

[9] Asthma is characterized by a chronic inflammation and narrowing of airway passages as well as acute flare-ups or asthma attacks, which are usually "triggered" by airway irritants, allergens, or infections.

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## Comment 2000 (continued)

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**1. Pre-2000 Scientific Studies Of The Health Effects Of Fine Particulate Matter.**

Fine particulate matter ("$PM_{2.5}$") consists of tiny particles less than 2.5 microns in diameter. Whereas coarse particulate matter ("$PM_{10\text{-}2.5}$"—particulates between 2.5 and 10 microns) is primarily composed of dusts and crustal elements from the earth in most locations, fine particulate matter is more likely to come from combustion sources, such as gasoline or diesel engines. $PM_{2.5}$ is a mixture of chemicals and metals that may be composed of liquids, solids, or both. Constituents of $PM_{2.5}$ may include acidic liquids, like sulfuric and nitric acids, organic chemicals including many of the air toxics, and tiny pieces of carbon soot. Because of its small size, $PM_{2.5}$ penetrates deeper into lung tissue than coarse particulate matter, even passing into the bloodstream.

EPA's review of the health effects of fine and coarse particulate matter in the mid-1990s led it to promulgate a new ambient air quality standard for fine particulate matter in 1997. See 40 CFR §50.7. This new standard was based on epidemiologic studies consistently showing that many of the health effects previously attributed to $PM_{10}$, such as increased mortality, hospitalization for respiratory problems, decreased lung function, and increased respiratory symptoms, were also associated with $PM_{2.5}$.[10] One study, for example, found that mortality was more strongly correlated with $PM_{2.5}$ than with $PM_{10}$, especially with respect to cardiovascular

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[10] Environmental Protection Agency, *Review of the National Ambient Air Quality Standards for Particulate Matter: Policy Assessment of Scientific and Technical Information*, EPA452-R-96-013 (1996).

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## Comment 2000 (continued)

Response Section in Chapter 35 ➥

and pulmonary causes of death.[11] In all, the EPA reviewed nine studies from the US and Canada and 11 from other parts of the world showing positive associations between fine particulate matter (or, in some cases, specific fine particulate constituents, such as sulfuric acids) and adverse cardiovascular and pulmonary health effects.[12] Based on these studies and other evidence of harm, EPA concluded --

> that fine particles are a better surrogate for those components of PM that are linked to mortality and morbidity effects at levels below the current standards [i.e., $PM_{10}$ NAAQS]. Moreover, a regulatory focus on fine particles would likely also result in controls on gaseous precursors of fine particles (e.g., SOx, NOx, VOC), which are all components of the complex mixture of air pollution that has most generally been associated with mortality and morbidity effects.

62 Fed.Reg. 38,667 (July 18, 1997). Given these findings two years before FHwA commenced the EIS, it had an obligation to consider this evidence when estimating, and disclosing to the public, the adverse health effects of emissions from the highway.

**2. The Recent Emergence Of Scientific Studies Of The Health Effects Of Mobile Source Air Toxics.**

A growing body of scientific studies has emerged showing a strong correlation between exposure to mobile source air toxics and a variety of health impacts.

[11] Schwartz, et al., *Is Daily Mortality Associated Specifically with Fine Particulates?*, 46(10) J. Air Waste Mgmt. Assoc. 927 (1996).

[12] Environmental Protection Agency, *Review of the National Ambient Air Quality Standards for Particulate Matter: Policy Assessment of Scientific and Technical Information*, EPA452-R-96-013 (1996) (see Table V-12).

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## Comment 2000 (continued)

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In December 2000, EPA evaluated mobile source emissions[13] and in March 2001 designated 21 chemicals as mobile source air toxics ("MSATs").[14] EPA selected these chemicals based on a scientific consensus that exposure to the chemicals pose serious threats to health, as reflected by their inclusion in EPA's Integrated Risk Information System ("IRIS") database.[15] To be listed in the IRIS database, a chemical must either be a known, probable, or possible carcinogen or cause significant non-cancer health effects, such as reproductive toxicity or neurotoxicity.

The chemical composition of the MSATs varies widely—ranging from metals to small organic compounds to dioxins—and their health impacts vary as well. Although significant information is available about the health effects of individual MSATs, less is known about the role they play compared to particulate matter, in part because of the difficulty of separately measuring the impact of each component of the toxic soup. A growing body of peer-reviewed scientific literature has identified serious health effects from short-term and long-term exposure to MSATs. Six of the MSATs come primarily from mobile sources (other MSATs have significant non-mobile sources). All six have extensive toxicologic data and many have substantial epidemiologic data documenting their health risks.

[13] Environmental Protection Agency, *Technical Support Document: Control of Emissions of Hazardous Air Pollutants from Motor Vehicles and Motor Vehicle Fuels*, EPA420-R-00-023 (December 2000).

[14] 66 Fed.Reg. 17,229-73 (Mar. 2001), citing *Technical Support Document*, see *id.* n.7.

[15] *Technical Support Document*, at 36.

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## Comment 2000 (continued)

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• *Acetaldehyde* is a probable human carcinogen based on studies in which rats and hamsters exposed to acetaldehyde formed nasal and laryngeal tumors, respectively.[16] It is also a potential developmental toxicant.[17] Further, exposure to acetaldehyde leads to irritation of the eyes, skin, and respiratory tract, indicating that it may contribute to worsening of health in people with asthma and other lung diseases.[18]

• *Acrolein* is a possible human carcinogen and a potent eye and respiratory-tract irritant. Animals chronically exposed to acrolein develop inflammation of the lungs and nasal passages.[19]

• *Benzene* is a known human carcinogen with extensive epidemiologic and toxicologic evidence that it causes leukemia.[20] In addition, benzene is toxic to bone marrow and blood cells, leading to decreased numbers of white and red blood cells.[21]

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[16] Environmental Protection Agency, *Integrated Risk Information System*, available at http://www.epa.gov/iris/subst/0419.htm#quaoral.

[17] Environmental Protection Agency, *Health Assessment Document for Acetaldehyde*, EPA/600/8-86-015A (1987).

[18] Environmental Protection Agency, *Technical Support Document: Control of Emissions of Hazardous Air Pollutants from Motor Vehicles and Motor Vehicle Fuels*, EPA420-R-00-023 (2000).

[19] Agency for Toxic Substance and Disease Registry, *Toxicological Profile for Acrolein* (1990), available at www.atsdr.cdc.gov/toxprofiles/tp124.html.

[20] Environmental Protection Agency, *Carcinogenic Effects of Benzene: An Update* (1998).

[21] Aksoy, *Hematotoxicity, Leukemogenicity and Carcinogenicity of Chronic Exposure to Benzene*, in E. Arinc, J.B. Schenkman, & E. Hodgson, eds., MOLECULAR ASPECTS OF MONOOXYGENASES AND BIOACTIVATION OF TOXIC COMPOUNDS pp. 415-34 (1991); Goldstein, *Benzene Toxicity*, 3 Occupational Medicine: State of the Art Reviews 541 (1998); Rothman, et

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## Comment 2000 (continued)

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• *1, 3-butadiene* is a known human carcinogen based on epidemiologic evidence. It also causes reproductive and developmental toxicity in animals exposed to long-term, low-level doses.[22]

• *Diesel particulate matter ("diesel PM") and diesel exhaust organic gases ("diesel EOG")* is a probable human carcinogen. There are several occupational epidemiologic studies associating diesel PM and EOG exposure with lung cancer, and EPA has estimated a range of cancer risk from a specific level of exposure.[23] The California Office of Environmental Health Hazard Assessment (OEHHA) conducted an independent review and determined a quantitative estimate of cancer risk that falls within the range of EPA estimates.[24]

Diesel PM and EOG also cause respiratory irritation and inflammation.[25] Further, a growing body of laboratory studies shows that exposure to diesel PM worsens allergic responses,

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al., *Hematotoxicity Among Chinese Workers Heavily Exposed to Benzene*, 29 Am. J. Ind. Med. 236 (1996).

[22] Environmental Protection Agency, *Health Risk Assessment of 1, 3-Butadiene*, EPA/600/P-8/001A (1998).

[23] See Environmental Protection Agency, *Health Assessment Document for Diesel Engine Exhaust*, EPA/600/8-90/057F (2002), available at http://cfpub2.epa.gov/ncea/cfm/recordisplay.cfm?deid=29060&CFID=474991&CFTOKEN=43362109 .

[24] California Office of Environmental Health Hazard Assessment, *Proposed Identification of Diesel Exhaust As a Toxic Air Contaminant: Health Risk Assessment for Diesel Exhaust* (1998), available at ftp://ftp.arb.ca.gov/carbis/regact/diesltac/partb.pdf.

[25] Pandya, et al., *Diesel Exhaust and Asthma: Hypotheses and Molecular Mechanisms of Action*, 110(Supp. 1) Environ. Health Perspect. 103 (2002).

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## Comment 2000 (continued)

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leading scientists to speculate that it may have a role in initiating allergic diseases, including asthma.[26] Lastly, diesel PM constitutes a significant portion of ambient fine particulate matter, which is associated with both acute and chronic cardiovascular toxicity and premature death. Recent reviews suggest that the combination of fine soot, acids, and other toxic chemicals associated with diesel PM leads to significant toxicity.[27]

• *Formaldehyde* is a potent eye and respiratory tract irritant that triggers asthma attacks and causes asthma-like symptoms in people without asthma.[28] EPA has classified formaldehyde as a probable human carcinogen, based on animal and human studies showing mainly nasal and upper respiratory cancers with exposure.[29]

While EPA's review of mobile source emissions focused on identifying the potential hazards associated with MSATs, the Multiple Air Toxics Exposure Study, conducted by the South Coast Air Quality Management District, provides pioneering, yet rigorous insight into the

[26] See, e.g., Nel, et al., *Enhancement of Allergic Inflammation by the Interaction Between Diesel Exhaust Particles and the Immune System*, 102(4 pt 1) J. Allergy Clin. Immunol. 539 (1998); Diaz-Sanchez, et al., *Diesel Exhaust Particles Directly Induce Activated Mast Cells to Degranulate and Increase Histamine Levels and Symptom Severity*, 106(6) J. Allergy Clin. Immunol. 1140 (2000); Diaz-Sanchez, et al., *Diesel Fumes and the Rising Prevalence of Atopy: An Urban Legend?*, 3(2) Curr. Allergy Asthma Rep. 146 (2003).

[27] Diesel Epidemiology Working Group, *Part 1: Report of the HEI Diesel Epidemiology Working Group* in RESEARCH DIRECTIONS TO IMPROVE ESTIMATES OF HUMAN EXPOSURE AND RISK FROM DIESEL EXHAUST (2002).

[28] Agency for Toxic Substance and Disease Registry, *Toxicological Profile for Formaldehyde*, available at www.atsdr.cdc.gov/toxprofiles/tp111.html.

[29] Enivronmental Protection Agency, *Integrated Risk Information System*, available at http://www.epa.gov/iris/subst/0419.htm#quaoral.

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magnitude of cancer risk associated with MSATs.[30] This landmark study, which relied on extensive monitoring data collected in and around the Los Angeles area, concluded that the overall cancer risk from air toxics to residents in the area was 1400 per one million, well over the 1:1,000,000 or 10:1,000,000 risk levels normally used by regulatory agencies. Using the estimate of diesel exhaust cancer risk from the California Office of Environmental Health Hazard Assessment, the MATES-II study found that 71% of overall cancer risk from air toxics in this area resulted from exposure to diesel exhaust emissions, 8% from 1, 3-butadiene, and 7% from benzene. Modeled exposure data for air toxics based on EPA's National Air Toxics Assessment reveal similar levels of cancer risk and demonstrate that diesel exhaust is the dominant source of that risk. For example, mobile sources contribute 96% of the cancer risk resulting from exposure to air toxics in Montgomery County, Maryland, where the proposed ICC is located, with diesel emissions responsible for 85% of the risk.[31]

**3. Epidemiologic Studies Of Health Effects And Mobile Source Emissions Show That MSATs and Particulate Matter Have A Significant Impact On Health.**

There is a strong body of indirect scientific evidence that exposure to mobile source air toxics has a substantial effect on human health. Most epidemiologic studies rely on exposure data for criteria air pollutants, such as $PM_{2.5}$ and $NO_x$, because the data for those pollutants are much more widely available. Because concentrations of air toxics from mobile sources are highly correlated with these criteria pollutants, health effects correlated with exposure to these

[30] SCAQMD, *Multiple Air Toxics Exposure Study: MATES II* (2000), available at http://www.aqmd.gov/matesiidf/matestoc.htm.

[31] Green Media Toolshed Scorecard (2006), available at http://www.scorecard.org/env-releases/hap/source-chemicals.tcl?geo_area_id=24031&geo_area_type=fips_county_code

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criteria pollutants are likely also to correlate with exposure to mobile source air toxics. This is particularly true with health effects known to be associated with particular air toxics, such as cancer and respiratory irritation.[32] Thus, to the extent epidemiologic studies use specific measurements of one or two constituents as proxies for the entire mixture of motor vehicle exhaust, the health effects correlated with these proxies may be correlated in part with the unmeasured air toxics.

***a. Correlation Between Asthma And Attending School Near A Major Roadway.***

Two studies specifically investigated the effects of motor vehicle emissions on children attending schools near major roadways. The first study assessed 2509 children from 24 schools located within 400 meters of a major roadway in the Netherlands. The study separately measured truck and car traffic, measured concentrations of $PM_{2.5}$, $NO_2$, and benzene on the school grounds, and took into account other factors that could cause allergic or respiratory problems, such as parental smoking. The study found that children going to school near roadways with heavy truck traffic were more likely to have allergies to outdoor pollens and to

[32] In this respect, motor vehicle emissions are similar to tobacco smoke, which is also a mixture of toxic gases and fine particulate matter. In each case, it is difficult to attribute specific toxicity to specific constituents. Delfino, *Epidemiologic Evidence for Asthma and Exposure to Air Toxics: Linkages Between Occupational, Indoor, and Community Air Pollution Research*, 110(Supp. 4) Environ. Health Perspect. 573, 586 (2002). The extensive epidemiologic literature on indoor environmental tobacco smoke does not rely on measurements of individual constituent chemicals within tobacco smoke, but instead uses substitute measures of exposure. Studies of the health effects of motor vehicle emissions do the same thing.

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## Comment 2000 (continued)

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have hyper-reactive airways, and that sensitization to asthma was correlated with $PM_{2.5}$ levels.[33]

The second study looked at 1019 children at 10 school sites in the San Francisco Bay Area. Five schools were located far from or upwind from major freeways, and five were located downwind and near freeways, with traffic loads ranging from 130,000 vehicles per day (i.e., less than the current traffic loads on US 95) to 230,000 vehicles per day, approximately the predicted traffic load on US 95. The study concluded that children attending schools with higher exposure to motor vehicle emissions had an increased risk of being diagnosed with asthma. Notably, significantly higher concentrations of black carbon (a measure of diesel PM and EOG) were measured at the schools downwind from the highways, and concentrations of $PM_{2.5}$ measured at the school located closest to a major freeway were 25% higher than measured at regional air quality monitoring stations (i.e., 15 $\mu g/m^3$ compared to 12 $\mu g.m^3$).[34]

***b. Correlation Between Respiratory Disease And Living Near A Major Roadway.***

Many studies have found a strong correlation between living near roads with high traffic and asthma. Not only do these studies show that exposure to mobile source emissions may

[33] Janssen, et al., *The Relationship Between Air Pollution from Heavy Traffic and Allergic Sensitization, Bronchial Hyperresponsiveness, and Respiratory Symptoms in Dutch Schoolchildren*, 111(12) Environ. Health Perspect. 1512 (2003).

[34] Kim, et al., *Traffic-Related Air Pollution Near Busy Roads: The East Bay Children's Respiratory Health Study*, 170(5) Am. J. Respir. Crit. Care Med. 520 (2004).

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## Comment 2000 (continued)

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trigger asthma attacks, a growing body of laboratory and epidemiologic literature suggests that mobile source emissions, especially diesel emissions, may play a role in initially causing asthma.

Studies have found a variety of asthma-related health effects correlated with exposure to motor vehicle pollution. One epidemiologic study of 16-year old Hispanic children living in areas of East Los Angeles with very high traffic density assessed the role of air toxics in worsening respiratory function. The study, which separately measured the effect of specific air toxics and criteria air pollutants, found positive correlations between asthma symptoms and air toxics, including benzene, acetaldehyde, diesel exhaust, and formaldehyde.[35]

Other studies show a strong correlation between exposure to mobile source emissions and asthma. A recent study from California showed that children living nearer freeways and with higher modeled exposures from freeway mobile source emissions had a higher risk of being diagnosed with asthma as well as higher medication use and wheezing.[36] One recent study showed higher rates of asthma in people exposed to mobile source emissions.[37] Another study, which followed a group of 3730 children from birth to two years of age and assessed each child's individual exposure to fine particulates and certain constituents of diesel exhaust, found significant correlations between exposure to motor vehicle emissions and upper

[35] Delfino, et al., *Asthma Symptoms in Hispanic Children and Daily Ambient Exposures to Toxic and Criteria Air Pollutants*, 111(4) Environ. Health Perspect. 647 (2003).

[36] Gauderman, et al., *Childhood asthma and exposure to traffic and nitrogen dioxide*, 16(6) Epidemiology 737 (2005).

[37] Kim, et al., *Traffic-Related Air Pollution Near Busy Roads: The East Bay Children's Respiratory Health Study*, 170(5) Am. J. Respir. Crit. Care Med. 520 (2004).

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## Comment 2000 (continued)

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respiratory infections.[38] A study involving 1068 Dutch schoolchildren found that children, especially girls, were more likely to be diagnosed with asthma and have respiratory symptoms if they lived within 100 meters of a major highway or if they had high exposure to truck traffic. The Dutch study also found a correlation between black carbon levels in school classrooms and respiratory symptoms in children living within 300 meters of a major roadway.[39] A study of children in Taiwan found that physician-diagnosed asthma was associated with traffic-related pollution.[40]

One hypothesis explaining the correlation between exposure to mobile source emissions and asthma is that diesel exhaust increases the risk of developing allergic disease. This hypothesis is supported by epidemiologic studies showing increased rates of allergic sensitization in children with higher exposure to mobile source emissions, especially truck traffic-related pollutants,[41] as well as by a growing body of laboratory evidence showing that components of diesel exhaust augment allergic responses to pollens and other allergens.[42]

[38] Brauer, et al., *Air Pollution from Traffic and the Development of Respiratory Infections and Asthmatic and Allergic Symptoms in Children*, 166(8) Am. J. Respir. Crit. Care Med. 1092 (2002).

[39] van Vliet, et al., *Motor Vehicle Exhaust and Chronic Respiratory Symptoms in Children Living Near Freeways*, 74(2) Environ. Res. 122 (1997).

[40] Guo, et al., *Climate, Traffic-Related Air Pollutants, and Asthma Prevalence in Middle-School Children in Taiwan*, 107(12) Environ. Health Perspect. 1001 (1999).

[41] Brauer, et al., *Air Pollution from Traffic and the Development of Respiratory Infections and Asthmatic and Allergic Symptoms in Children*. 166(8) Am. J. Respir. Crit. Care Med. 1092 (2002); Janssen, et al., *The Relationship Between Air Pollution from Heavy Traffic and Allergic*

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## Comment 2000 (continued)

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Epidemiologic studies have consistently shown that people with higher exposures to roadway air pollutants have more hospitalizations for asthma, more respiratory symptoms, and poorer lung function. A review of 20 studies published between 1993 and 2000, found all but one showed that higher exposures to roadway pollutants, especially heavy-truck exhaust, were correlated with worsened asthma, decreased lung function, and more symptoms of asthma.[43] Subsequent studies have confirmed this correlation. For example, a 2001 study showed that exposure to moderate traffic pollution was associated with increased inflammatory markers and decreased lung function in children.[44] A study in Roxbury, Massachusetts, found that exposure to fine particulate matter and polycyclic aromatic hydrocarbons (a constituent of

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*Sensitization, Bronchial Hyperresponsiveness, and Respiratory Symptoms in Dutch Schoolchildren*, 111(12) Environ. Health Perspect. 1512 (2003); Wyler, et al., *Exposure to Motor Vehicle Traffic and Allergic Sensitization*, 11(4) Epidemiology 450 (2000).

[42] Nel, et al., *Enhancement of Allergic Inflammation by the Interaction Between Diesel Exhaust Particles and the Immune System*, 102(4 pt 1) J. Allergy Clin. Immunol. 539 (1998); Diaz-Sanchez, et al., *Diesel Exhaust Particles Directly Induce Activated Mast Cells to Degranulate and Increase Histamine Levels and Symptom Severity*, 106(6) J. Allergy Clin. Immunol. 1140 (2000); Diaz-Sanchez, et al., *Diesel Fumes and the Rising Prevalence of Atopy: An Urban Legend?*, 3(2) Curr. Allergy Asthma Rep. 146 (2003).

[43] Delfino, *Epidemiologic Evidence for Asthma and Exposure to Air Toxics: Linkages between Occupational, Indoor, and Community Air Pollution Research*, Environ. 110(Supp. 4) Environ. Health Perspect. 573 (2002).

[44] Steerenberg, et al., *Traffic-Related Air Pollution Affects Peak Expiratory Flow, Exhaled Nitric Oxide, and Inflammatory Nasal Markers*, 56(2) Arch. Environ. Health 167 (2001).

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## Comment 2000 (continued)

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diesel emissions) was associated with asthma hospitalizations.[45] A study from Nottingham, United Kingdom, concluded that living within 90 meters of main roads correlated with an increased risk of wheezing illness in children age 4-11.[46] A study in Munich, Germany, demonstrated that the mobile source emissions of particulate matter and nitrogen dioxide were associated with symptoms such as dry cough at night and cough without infection in children ages 1 and 2.[47] A study in East and West Germany found that during a time period of declining pollution in East Germany (1991-2000), improvements in lung function seen in 5-7 year old children over that time were diminished in children living within 50 meters of a busy roadway.[48] A study in Buffalo, New York, showed that the risk of asthma hospitalization increased with exposure to motor-vehicle emissions.[49] A study in Southeast Toronto demonstrated that the risk of hospital admission for asthma, bronchitis, chronic obstructive

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[45] Levy, et al., *Fine Particulate Matter and Polycyclic Aromatic Hydrocarbon Concentration Patterns in Roxbury, Massachusetts: A Community-Based GIS Analysis*, Environ. 109(4) Environ. Health Perspect. 341 (2001).

[46] Venn, et al., *Living Near a Main Road and the Risk of Wheezing Illness in Children*, 164(12) Am. J. Respir. Crit. Care Med. 2177 (2001).

[47] Gehring, et al., *Traffic-Related Air Pollution and Respiratory Health During the First 2 Years of Life*, 19(4) Eur. Respir. J. 690 (2002).

[48] Sugiri et al., *The influence of large-scale airborne particle decline and traffic-related exposure on children's lung function*, 144(2) Environmental Health Perspectives 282 (2006).

[49] Lin, et al., *Childhood Asthma Hospitalization and Residential Exposure to State Route Traffic*, 88(2) Environ. Res. 73 (2002).

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pulmonary disease (i.e., emphysema and chronic bronchitis), pneumonia, and upper respiratory tract infection increased with increased exposure to $PM_{2.5}$.[50] A 2002 study in 14 cities also associated increased hospital admissions for chronic obstructive pulmonary disease, heart disease, and pneumonia with particulate matter from motor vehicles.[51] A 2005 study from Germany found 55 year old women living near roadways had a higher risk of developing COPD and having decreased lung function.[52]

***c. Association Between Lung Cancer And Living Near A Roadway.***

Two studies of individuals living near roadways show a correlation between traffic density and lung cancer. A 2003 study found excess lung cancer risks associated with living near roads.[53] A study in Stockholm found a 40% increase in lung cancer risk for the highest group of average traffic-related $NO_2$ exposure.[54] Because $NO_2$ generally is not associated with

[50] Buckeridge, et al., *Effect of Motor Vehicle Emissions on Respiratory Health in an Urban Area*, 110(3) Environ. Health Perspect. 293 (2002).

[51] Janssen, et al., *Air Conditioning and Source-Specific Particles as Modifiers of the Effect of $PM_{10}$ on Hospital Admissions for Heart and Lung Disease*, 110 Environ. Health Perspect. 43 (2002).

[52] Schikowski, et al. *Long-term air pollution exposure and living close to busy roads are associated with COPD in women*. 6(1) Respiratory Research 152 (2005).

[53] Nafstad, et al., *Lung Cancer and Air Pollution: A 27 Year Follow up of 16,209 Norwegian Men*, 58(12) Thorax 1071 (2003).

[54] Nyberg, et al., *Urban Air Pollution and Lung Cancer in Stockholm*, 11(5) Epidemiology 487 (2000).

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lung cancer, it is likely the correlation reflects exposure to carcinogenic motor vehicle emissions, such as diesel particulates and other air toxics.

***d. Association Between Adverse Reproductive Effects And Exposure To Motor Vehicle Pollutants.***

One study demonstrated that long-term exposure to motor vehicle pollutants are correlated with low birth weight and pre-term birth.[55]

**C. Methods Are Available Methods To Assess Health Impacts From Fine Particulate Matter And Mobile Source Air Toxics.**

The ICC FEIS indicates that an ICC build alternative will increase MSAT emissions in the ICC study area by one to six percent in 2030 relative to a non-build alternative, due primarily to increased VMT for the build alternative.[56] However, the ICC FEIS does not evaluate localized MSAT emissions and concentrations that can be anticipated to result in pollution hot spots close to the highway, especially in the early years of operation of the ICC, from 2010 onward, when MSAT emissions can be anticipated to be at their highest levels. FHWA asserts it is unable to evaluate localized emission concentrations or health effects because of uncertainties in the MOBILE6.2 model, especially with respect to diesel particulate matter, and uncertainties surrounding the health effects of MSAT pollutants.[57] However, exposure and risk assessment

[55] Wilhelm and Ritz, *Residential Proximity to Traffic and Adverse Birth Outcomes in Los Angeles County, California, 1994-1996*, 111 Environ. Health Perspect. 207 (2003).

[56] Intercounty Connector Final Environmental Impact Study, FHWA, Page IV-326 (2006).
[57] Intercounty Connector Final Environmental Impact Study, FHWA, Page IV-328 (2006).

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tools are available to establish the degree of risk roadside populations face from exposure to fine particulate matter and air toxic emissions of motor vehicles and to determine diesel particulate emissions.

Two different methods are available to assess the risks to human health from particulate matter and mobile source air toxics. Both methods combine estimates of exposure with estimates of the "dose-response" function to produce an estimate of risk associated with that exposure.[58] One method is based on epidemiologic data that establishes how the risk of particular health effects changes with exposure to particular pollutants.[59] If epidemiologic data are insufficient (which is the case for many carcinogens), the dose-response function can be obtained from toxicologic experiments measuring the dose-response for rodents, with the results extrapolated to humans and real world exposures.

Based on epidemiologic data for fine particulate matter, EPA has estimated dose-response functions for a large number of health effects, including total mortality, hospitalizations for heart disease and lung disease, hospitalizations for asthma in children, and asthma attacks in children. For example, EPA's regulatory impact analysis for heavy duty diesel standards calculated the change in 13 health effects as a result of reductions in $PM_{2.5}$ emissions

[58] The "dose-response" is an estimate of the risk of a specific health effect in response to a specified dosage, or exposure, of the pollutant.

[59] Using epidemiologic data, one can calculate the "relative risk"—the increase in risk in a "real-world" human population from a measured exposure. When the relative risk is combined with the baseline frequency of the health effect (i.e., in the absence of exposure to the pollutant), one can calculate the increase in the number of cases in response to the increase in exposure.

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from diesel engines because of the new standard.[60] EPA's 1996 criteria document for particulate matter, which was the basis for the 1997 air quality standard for $PM_{2.5}$, assessed four endpoints (short and long-term mortality, hospital admissions for all respiratory causes, and respiratory symptoms) using concentration-response parameters derived from epidemiologic studies.[61] Similar concentration response parameters could be established for projected changes in $PM_{2.5}$ due to the construction of the Intercounty Connector, yielding estimates of changes in health effects for individuals in affected neighborhoods.

Cancer risks from exposure to MSATs may be determined using a methodology similar to that used in the MATES-II study. Changes in the concentrations of the six priority MSATs may be estimated using EPA's MOBILE6.2 model.[62] For those MSATs with cancer unit risk values in EPA's IRIS database, the estimated concentrations of the individual air toxics from the construction of the Intercounty Connector can be combined with the cancer unit risk values to produce estimates of cancer risk from exposure to individual air toxics as well as the total risk from exposure to all toxics combined.

[60] Environmental Protection Agency, *Heavy-Duty Standards/Diesel Fuel RIA* - EPA420-R-00-026 (Dec. 2000), Table VII-14, p. VII-42

[61] Environmental Protection Agency, *Review of the National Ambient Air Quality Standards for Particulate Matter: Policy Assessment of Scientific and Technical Information*, EPA452-R-96-013 (1996), Table VI-2, p. VI-13

[62] Environmental Protection Agency, *User's Guide to MOBILE6.1 and MOBILE6.2 Mobile Source Emission Factor Model*, EPA420-R-03-010 (Aug. 2003), p.16

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The ICC FEIS argues that because large reductions in MSATs are expected by 2030 for all alternatives, FHWA does not believe that there will be significant adverse impacts on the human environment from MSATs as a result of the ICC.[63] Moreover, they note that uncertainties associated with the absolute emission estimates and difficulty assessing exposure at the project level and associated health impacts complicate health impact assessment. Thus, they argue they have no obligation to consider cancer risks that may decrease even if traffic increases. The argument is misguided. Health risks to populations in 2030, especially cancer risks, will be based on exposures from many years prior to 2030. Because old vehicles are not immediately removed from service, aggregate emissions from new cleaner vehicles and the pre-2007 vehicles likely will continue to increase for a decade or more before total emissions begin to decline. Thus cancer risks must be modeled on emissions characteristics that will be prevalent from 2010 to 2030, not just those that become prevalent in 2030. Moreover, acute and chronic cardiovascular and respiratory health risks will be significant impacts for communities adjacent to a new freeway from its construction on. It would be far more appropriate to estimate cumulative risks as of 2030 rather than assuming no risks prior to 2030, and then discounting them because of expected declines in mobile source air toxic emissions.

**IV. CONCLUSION**

Based on the foregoing reasons and authorities, the ICC FEIS should not be approved. Instead, FHWA should prepare a supplemental EIS evaluating the localized impacts of fine

[63] Intercounty Connector Final Environmental Impact Study, FHWA, Page IV-328 (2006).

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particulate matter for people living in close proximity to the proposed highway and other highways in the area that will experience increased traffic as a result of the ICC. This supplemental EIS should also evaluate the impacts of mobile source air toxics.

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**SUMMARY OF NEW SCIENTIFIC LITERATURE DOCUMENTING ADVERSE HEALTH EFFECTS IN PEOPLE EXPOSED TO HIGH-TRAFFIC ROADWAYS**

**John Balbus, MD, MPH**
**Environmental Defense**
**May 24, 2007**

**Introduction**

Studies published in the scientific literature since March 2006 strengthen the evidence for harm to health from traffic-associated air pollution and extend our understanding of the nature of roadside exposures and the special susceptibility of children, the elderly, and those with underlying diseases. These studies indicate that exposure to the mixture of toxic pollutants coming from motor vehicles, even in regions that are considered to be in attainment of federal air quality standards, can worsen asthma, impair lung development, and contribute to heart disease and premature death. These effects are especially apparent in children, who have heightened susceptibility to traffic-associated air pollution because of their smaller size, increased respiratory rates, actively developing lungs, and greater and more active time spent outdoors. The studies indicate a zone that extends from approximately 500 to 1500 feet around major roads that contains elevated levels of traffic-related pollutants and describe increased risks of adverse health effects for people living or going to school inside this zone. While not all studies explicitly link actual traffic counts to exposure levels, several studies below associate elevated exposures with traffic counts as low as 10,000 cars per day. This report summarizes recent publications and other information relevant to determining the public health impact of population exposures to traffic-related air pollution.

As indicated below, the U.S. EPA notes over 1000 chemicals in the mixture of air pollutants emitted by motor vehicles, of which four are criteria air pollutants and 93 are toxic chemicals appearing in EPA's IRIS database due to carcinogenic or other well-documented health effects. While many of the studies cited below measure specific air pollutants as indicators of traffic-related pollutants, the majority of the studies associate adverse health effects with proximity to traffic-related sources of air pollution and do not implicate specific air pollutants as the sole or even dominant contributor to adverse health effects. In fact, because of similarities in toxic effects and toxic mechanisms, it is likely that many of the traffic-related pollutants known to be harmful, as evidenced by their inclusion in the IRIS database, jointly contribute to the observed adverse health effects.

***Exposure-related studies***

A recently published meta-analysis from the Harvard School of Public Health reviewed studies of pollution concentrations near roadways. The authors conclude that the spatial extent of significantly elevated levels depends on the type of air pollution, with ultrafine particle counts elevated as far out as 300 meters, elemental carbon or fine particulate mass elevated as far out as 400 meters, and nitrogen dioxide elevated as far out as 500 meters.[1]

In Amsterdam, researchers have also assessed outdoor and personal exposure to traffic-related air pollution among children living on streets with varying degrees of traffic intensity. The authors of a 2006 study monitored children aged 9-12 years who were exposed to soot and NOx and measured indoor/outdoor NOx levels at their homes and schools. Results demonstrate that children living near busy roads had 35 percent higher personal exposure to soot than those who lived at an urban background location, even when they attended schools away from busy roads. In this study, a

[1] Zhou Y, Levy JI. Factors influencing the spatial extent of mobile source air pollution impacts: A meta-analysis. BMC Public Health, in press.

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busy road was defined as one having average daily traffic of more than 10,000 cars. The study supports using "living near a busy road" as a measure of exposure in epidemiological studies on the effects of traffic-related pollution in children.[2]

In 2006, the EPA released an updated master list of more than one thousand chemicals emitted by mobile sources.[3] This list includes, in Table 4, ninety-three chemicals emitted by mobile sources that are also in EPA's IRIS database and are well-recognized to have serious health effects from environmental levels of exposure, including cancer, respiratory irritation, and neurotoxicity. The large number of chemicals on this list underscores the complexity of the mixture of air pollutants to which people near roadways are exposed and the many opportunities for synergistic effects of similarly acting chemicals.

***Studies of health effects in children***

Several studies published recently have demonstrated serious effects of motor vehicle emissions on children living near roadways. A 2007 study published in the *European Respiratory Journal* looked at the relationship between traffic-associated air pollutants and the development of asthma, allergy, and infections in children during the first four years of life. The authors followed 4,000 children in the Netherlands, analyzing data on self-reported wheeze, dry-night-time cough, ear/nose/throat infections, skin rash and physician diagnoses of asthma, bronchitis, influenza and eczema. They found a positive association between traffic-related pollution and respiratory infections as well as certain measures of asthma and allergy.[4]

A 2007 *Lancet* study from California documented that both regional elevations of air pollution and local exposure to freeway traffic have harmful, independent effects on children's lung development. The authors followed 3,677 children living in 12 southern California communities with varying air quality over a period of eight years, recording annual lung-function measurements and identifying indicators of residential exposure to freeway traffic. Results from eight years of followup indicate that children living within 500 meters of freeways have substantial deficits in lung growth and development and pulmonary function compared with those living at least 1500 meters from freeways.[5] Subsequent testimony from the lead author on this study indicates that the effects were seen in association with exposure to pollutants from freeways with average daily traffic levels as low as 45,000 vehicles.[6]

A third study, published in 2006, examined the relationship between local traffic-related exposure and asthma and wheeze in children in southern California, ages 5-7 years. The authors assessed residential exposure by proximity to a major road and modeled exposure to local traffic-related pollutants. They found an association between living within 75 meters of a major road and increased risk of lifetime asthma, prevalent asthma, and wheeze, and determined that the effect of

[2] Van Roosbroeck et al. Long-term personal exposure to traffic-related air pollution among school children: a validation study. *Science of the Total Environment*, September 2006, 338(2-3):565-573.
[3] US EPA. Office of Air and Radiation. Expanding and Updating the Master List of Compounds Emitted by Mobile Sources - Phase III. Final Report. EPA420-R-06-005.
[4] Brauer M et al. Air pollution and development of asthma, allergy and infections in a birth cohort. *European Respiratory Journal* 2007, 29(5): 879-888.
[5] Gauderman WJ et al. Effect of exposure to traffic on lung development from 10 to 18 years of age: a cohort study. *The Lancet*, February 2007, 369(9561): 571-577.
[6] Gauderman WJ, Written responses to questions posed by members of the Colorado legislature, submitted to Colorado House Education Committee hearing, March 5, 2007. Text appended to this report.

## Comment 2000 (continued)

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residential proximity to roadways was more pronounced in girls. The authors conclude that living near a major road is associated with asthma.[7]

A study in 2007 estimated long-term exposure to traffic-related air pollutants and assessed adverse health effects, collecting particulate matter measurements at 40 sites in Munich, Germany. The authors found that estimated $PM_{2.5}$ exposures, $PM_{2.5}$ absorbance, and $NO_2$ were 12.8 $\mu g/m^3$, $1.7x10^{-5}$ $m^{-1}$, and 35.3 $\mu g/m^3$ respectively. There were significant associations between $PM_{2.5}$ and symptoms including sneezing, runny/stuffed nose during the first year of life, as well as between $NO_2$ and dry cough at night during the first year of life. Living within 50 meters of busy roads increased the risk of wheezing and asthmatic/spastic/obstructive bronchitis. In this study, a "busy" road was defined as one with traffic greater than 3000 automobiles per day.[8]

Lastly, a UCLA Health Policy Research Brief described a study from Southern California that found that children with asthma, especially in low-income groups, had three-fold higher rates of emergency room visits and more severe asthma exacerbations when exposed to high traffic density compared to low traffic density. Traffic density was estimated by multiplying average daily traffic counts within a 500 foot buffer zone around the residence by the miles of road segments within that zone, then dividing by the area. Effects were seen at the medium traffic density level (20,000-200,000 vehicle miles traveled per square mile) as well as the high traffic density level. The authors conclude that "further reduction of traffic related air pollution is needed to reduce the burden of asthma, especially among low-income and racial/ethnic minority groups."[9]

***Studies in other susceptible subpopulations***

Other recent studies have examined the effect of traffic-related air pollution on specific populations aside from children, such as women or the elderly. A study from Worcester, MA, published in 2007 in *Environmental Health Perspectives*, found a 4-5% increase in the risk of acute myocardial infarction (heart attack) among men and women who were exposed long-term to greater amounts of vehicle traffic or lived near major roadways. The risks were highest among those less than 65 years of age.[10] Another 2007 study published in the *New England Journal of Medicine* followed 65,893 postmenopausal women without history of cardiovascular disease in 36 U.S. cities over the course of six years. The authors found an association between long-term exposure to fine particulate air pollution and the incidence of cardiovascular disease and death among this population.[11] While this study did not specifically measure traffic-related particulate exposures, it complements previous studies of traffic-related particulate matter effects on the heart and strengthens the findings of an association in women.

One 2006 study looking at the association between hospital admissions for respiratory disease among the elderly and traffic intensity near the homes of the elderly in Montreal found that increased odds of being hospitalized for a respiratory versus control diagnosis were associated with

[7] McConnell et al. Traffic, susceptibility, and childhood asthma. *Environmental Health Perspectives*, May 2006, 114(5):766-772.
[8] Morgenstern et al. Respiratory health and individual estimated exposure to traffic-related air pollutants in a cohort of young children. *Occupational and Environmental Medicine*, January 2007, 64(1): 8-16.
[9] Meng YY, Rull RP, Wilhelm M, Ritz B, English P, Yu H, Nathan S, Kuruvilla M and Brown ER. Living Near Heavy Traffic Increases Asthma Severity. Los Angeles: UCLA Center for Health Policy Research, 2006.
[10] Tonne C, Melly S, Mittleman M, Coull B, Goldberg R, Schwartz J. A case-control analysis of exposure to traffic and acute myocardial infarction. *Environmental Health Perspectives*. 2007 Jan;115(1):53-7.
[11] Miller KA et al. Long-term exposure to air pollution and incidence of cardiovascular events in women. *New England Journal of Medicine*, February 2007, 356(5):447-458.

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higher road traffic levels near patients' homes, even after adjusting for the appraised value of those homes. The study's findings suggest that road traffic intensity might have an effect on the respiratory health of elderly residents, and this association is not just a reflection of socioeconomic status. Road traffic intensity was measured as the amount of estimated traffic during the 3 hour peak. The cutoff between medium and high intensity was 3160 vehicles per 3 hour peak. Effects were seen with both medium and high intensity traffic exposures.[12]

***Conclusions***

In summary, new scientific studies published since 2006 provide more robust evidence for serious health effects from exposure to traffic-related air pollution. The studies indicate that within a 500-1500 foot zone around major roadways, people are exposed to elevated levels of a complex mixture of air pollutants, many of which are known to cause significant health risks. The use of indicator pollutants to assess exposures is necessary but should not be interpreted as demonstrating that observed health effects are related only to exposures to the indicators. It is biologically plausible that exposure to the complex mixture of traffic-related pollution is more harmful than exposure to only one or two of the primary constituents of the mixture. The studies also suggest that health risks are elevated at traffic counts in the thousands and low ten thousands of vehicles per day. Strengths of the new studies include the fact that several involve following cohorts of children over time, which provides more certainty in the diagnosis of asthma and other conditions, and improved exposure assessments.[13] Taken together, these studies strongly suggest that complying with regional federal air standards under the Clean Air Act is not sufficient to protect public health. Federal agencies must provide greater protection for populations exposed to traffic-related pollution from major roadways.

[12] Smargiassi A et al. Traffic intensity, dwelling value, and hospital admissions for respiratory disease among the elderly in Montreal (Canada): a case-control analysis. *Journal of Epidemiology and Community Health*, 2006, 60:507-512.
[13] See, for example, Jerrett M. Does traffic-related air pollution contribute to respiratory disease formation in children? Eur Respir J. 2007 May;29(5):825-6.

## Comment 2001

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35.2.3A

Print View Page 1 of 1

From: Jim STRUVE <jimstruve@mac.com>
To: <mountainview@utah.gov>
Date: Thursday - November 15, 2007 7:08 AM
Subject: Mountain View Corridor Feedback

Dear Sir/Maam,

I am a resident of Salt Lake City. I have been reading with interest the plans for the Mountain View Corridor highway. I am very concerned about the continued emphasis on building new highways rather than increasing the infra structures for public transportation. I believe that building an extensive and efficient public transportation network (TRAX, buses, bike corridors, etc.) will better serve the needs of the Salt Lake Metropolitan communities in the long run. I believe there is strong public support to move away from expensive investments in building new highways.

If new roads are to be built, please do not place such roadways so close to schools, homes, etc.

Thank you for your consideration of my input.

Jim Struve
722 East 900 South
Salt Lake City, UT. 84105
jimstruve@mac.com

https://email.udot.utah.gov/gw/webacc?User.context=mx9nq0Sn0um9hseFmf&Item.drn=261z18... 11/29/2007